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DEVELOPMENT AND CONSENSUS PROCESS

The development of this document involved a process of text review by the expert working group and revision by the authors. It culminated in consensus as indicated by sign off from each working group member and author.
Pressure, shear, friction and microclimate in context

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The overall goal of clinical care is to restore or maintain health. Unfortunately, however, iatrogenic injuries sometimes occur. Although not all pressure ulcers are iatrogenic, most are preventable. Pressure ulcers are one of the most frequently reported iatrogenic injuries in developed countries. Inappropriate care methods, such as leaving vulnerable patients in potentially damaging positions for long periods of time, or massaging reddened areas of skin, often remain in practice long after evidence has shown them to be harmful or ineffective. Education is critical in ensuring that all members of a clinical team act to prevent and treat pressure ulcers according to the best evidence available.

The most recent definition of pressure ulcers, which has been produced by an international collaboration of the National Pressure Ulcer Advisory Panel (NPUAP) and the European Pressure Ulcer Advisory Panel (EPUAP), highlights current understanding of the role of extrinsic factors in the development of pressure ulcers1,2 (Box 1). Pressure, which is often related to decreased mobility, has long been viewed as the most important extrinsic factor in pressure ulcer development. However, recent and ongoing research is revealing that shear, friction and microclimate also have important roles, and that there are significant and complex relationships between all of the extrinsic factors. For example, pressure and shear are closely linked, friction has a role in the development of shear, and microclimate influences the susceptibility of skin and soft tissues to the effects of pressure, shear and friction.

The concepts involved in understanding pressure, shear, friction and microclimate and their synergistic actions in the formation of pressure ulcers are complex. Consequently, the expert working group involved in producing Pressure ulcer prevention: prevalence and incidence in context1 proposed a new document to aid understanding of these extrinsic factors. The expert working group decided that, even though pressure, shear, friction and microclimate are inextricably inter-related, this new project would tackle each extrinsic factor individually with the aim of building understanding of the physics involved. This understanding should enable clinicians to better comprehend developments in the field and, most importantly, will underpin effective and consistent implementation of pressure ulcer prevention protocols.

The three papers – Pressure in context, Shear and friction in context, and Microclimate in context – follow a similar structure. They start by defining the relevant extrinsic factors and how individually they contribute to the aetiology of pressure ulcers. The relationships between the factors are explained and emphasised, and the evidence for the role of the factors in the development of pressure ulcers is discussed. The latter sections of the three papers describe how patients at risk from each extrinsic factor can be identified. The papers then explain the types of and rationale for the clinical interventions that aim to prevent or ameliorate the adverse effects of each of the extrinsic factors discussed. It should be noted that, although the document covers many major facets of pressure ulcer prevention, discussion of comprehensive prevention protocols is beyond its scope.

Much research remains to be undertaken to further develop our understanding of the intrinsic and extrinsic causes of pressure ulcers. But as this document shows, there are some important underlying principles for preventing pressure ulcers resulting from the extrinsic factors of pressure, shear, friction and microclimate. All clinicians should understand these principles and implement them in their daily practice.

REFERENCES

BOX 1 New NPUAP/EPUAP definition of pressure ulcers

“A pressure ulcer is localized injury to the skin and/or underlying tissue, usually over a bony prominence, as a result of pressure, or pressure in combination with shear. A number of contributing or confounding factors are also associated with pressure ulcers; the significance of these factors has yet to be elucidated.”

PRESSURE, SHEAR, FRICTION AND MICROCLIMATE IN CONTEXT | 1
INTRODUCTION
Pressure has been recognised as the most important extrinsic factor involved in the development of pressure ulcers for many years. Consequently, it features prominently in definitions of pressure ulcers, including the recent definition produced by the National Pressure Ulcer Advisory Panel (NPUAP) and European Pressure Ulcer Advisory Panel (EPUAP)1,2.

This paper explains what pressure is, how pressure contributes to pressure ulcer formation and how to identify patients at risk of injury from pressure. It then describes the rationale and mode of action of interventions that reduce the magnitude and duration of pressure and, consequently, the risk of pressure ulcer development.

WHAT IS PRESSURE?
Pressure is defined as the amount of force applied perpendicular to a surface per unit area of application.

A force applied over a small area will produce greater pressure than the same force applied over a larger area (Figure 1). The unit of force is the newton (N). The unit of pressure is newtons per square metre (N/m²), pascals (Pa) or millimetres of mercury (mmHg).

In addition to the perpendicular force that is involved in pressure, forces may be applied parallel to the skin surface (Figure 2). These are shear forces and contribute to shear stresses, which are also measured in terms of force per unit area (see: Shear and friction in context3, pages 11-18). Stress is a generic name for effects that are defined in terms of force per unit area of application.

Pathophysiology of pressure damage
Skin that has been subjected to potentially damaging levels of pressure initially appears pale from reduced blood flow and inadequate oxygenation (ischaemia). When the pressure is relieved, the skin quickly becomes red due to a physiological response called reactive hyperaemia. If the ischaemia has been sufficiently short lived, blood flow and skin colour will eventually return to normal.

More prolonged ischaemia can cause blood cells to aggregate and block capillaries, perpetuating the ischaemia. Capillary walls can also become damaged, allowing red blood cells and fluid to...
leak into the interstitial space. This process results in the non-blanchable erythema, skin discolouration and induration that are seen with Category/Stage I pressure ulcers. Continued ischaemia results in necrosis of the skin and underlying tissue, and the superficial and deeper tissue breakdown seen with higher category/stage pressure ulcers. High pressure is also known to physically damage muscle tissue by deforming and rupturing muscle cells.

Deep tissue injury
The new NPUAP/EPUAP pressure ulcer classification contains an additional category for use in the USA: deep tissue injuries. Clinical experience suggests that these usually present with purple skin around 48 hours following a pressure event, e.g. being unconscious on the floor, and become necrotic quickly, even when care is provided (Figure 4).

**WHAT DO WE KNOW ABOUT PRESSURE AND PRESSURE ULCERS?**
Because the primary mechanism of pressure-induced tissue damage is thought to be blood flow reduction, papers that discuss pressure ulcers frequently mention research done in the 1930s by Landis. This work found that the pressure in the arteriolar limb of a capillary in the human finger was on average 32mmHg. This value was then mistakenly generalised to be the pressure required to compress capillaries to prevent blood flow (the capillary closing pressure), and the pressure below which pressure redistributing devices aimed to reduce interface pressure. However, many following studies also demonstrated a wide range of pressure in capillaries at various anatomical locations, with values dependent on age and concomitant disease.

**Relationship between duration and intensity of pressure**
By the middle of the 20th century, duration of pressure was suspected to be a factor in pressure ulcer development, but quantitative data were missing until Kosiak started to publish his experiments in 1959. These involved loading tissues with known pressures for specific durations. Histological examination was used to assess tissue viability.

Kosiak reported a relationship between amount of pressure, duration of application and the development of tissue damage in canine and rat experiments. He stated that, "microscopic pathologic changes were noted in tissues subjected to as little as 60mmHg for only one hour." Kosiak reported a relationship between amount of pressure, duration of application and the development of tissue damage in canine and rat experiments. He stated that, "microscopic pathologic changes were noted in tissues subjected to as little as 60mmHg for only one hour."
Figure 5: Proposed modification to Reswick and Rogers pressure-time curve (adapted from [15–17]).

The area above the curves represents durations and intensities of pressure that are unlikely to result in tissue damage; the area below the curves represents durations and intensities of pressure that are likely to result in tissue damage. The area above the curves represents durations and intensities of pressure that are unlikely to result in tissue damage; the area below the curves represents durations and intensities of pressure that are likely to result in tissue damage; the area below the curves represents durations and intensities of pressure that are likely to result in tissue damage; the area below the curves represents durations and intensities of pressure that are likely to result in tissue damage; the area below the curves represents durations and intensities of pressure that are likely to result in tissue damage.

Recently, it has been proposed that the Reswick and Rogers curve be modified to reflect more recent animal studies and clinical experience that high pressures can cause pressure damage within a relatively short time, but that lower pressures can be applied for long periods without damage occurring [15–17] (Figure 5).

Pressure and temperature
The effects of pressure may be modulated by skin temperature. Work by Kokate et al and Iaizzo et al in pigs concluded that skin and soft tissue damage due to pressure could be reduced by localised skin cooling [16–19] (see: Microclimate in context [20], pages 19–25).

Physiological effects
In an experiment to measure the effects of pressure on blood flow in the human forearm, pressure ranging from 0 to 175 mmHg was applied to the skin. The results showed that blood flow was affected by pressure on the skin to a greater extent in a deep artery than in a skin capillary [20]. Future investigations to measure deep tissue blood flow may contribute to understanding of the ischaemic factors in the mechanism of pressure ulcer formation.

How can internal stresses be measured?
Many studies investigating the role of pressure in the development of pressure ulcers measure pressure at the skin surface (interface pressure). Even so, bioengineering work carried out since the 1980s has indicated that internal tissue stresses cannot be predicted by means of interface pressure measurements [15,14]. Stresses within tissues measured in an animal model demonstrated that pressure is three to five times higher internally near a bony prominence than the pressure applied to the skin over the prominence [22]. Computer modelling has confirmed that the highest stresses are near the bony prominence [23].

Identifying patients at risk from pressure
Patients at highest risk from pressure are those in whom pressure on skin would go unrelieved if the healthcare staff did not move them in a bed or chair. Asking the question, "Can the patient feel pressure and move about or ask others to move him?" is an important first step. When the answer to the question is, "No"," high risk patients can be identified quickly by all staff. General patient assessment will indicate other factors, eg reduced tissue perfusion or poor nutrition, which may make a patient more vulnerable to the effects of pressure. Some of these factors increase risk by amplifying the effects of shear and friction, or by reducing skin and tissue tolerance to pressure (see: Shear and friction in context [3], pages 11–18 and Microclimate in context [20], pages 19–25).

Several tools are available for assessing overall risk of pressure ulcer development; these are based on a number of factors, including pressure [24–26]. Although there are limitations to the use of such tools and alternative approaches have been suggested [27], risk assessment tools are highly valued in clinical practice.

Reducing risk from pressure
Best practice care of patients at risk of pressure ulceration has numerous facets that aim to ameliorate the effects of intrinsic risk factors (such as poor nutrition, concomitant disease, dry skin) and extrinsic factors (such as shear and friction, or incontinence). (See: Shear and friction in context [3], pages 11–18 and Microclimate in context [20], pages 19–25.)

With respect to pressure, efforts centre on reducing or removing the pressure applied to the skin of vulnerable patients. The principles involved also apply to patients with existing pressure damage. Patients should avoid sitting or lying on areas of non-blanchable erythema or pressure ulcers. If such areas or wounds fail
to improve or deteriorate, practitioners must consider whether continued pressure over the area is contributing to the problem. Clinical judgement is essential in determining how best to provide care for patients at risk from damage by pressure.

PRESSURE REDISTRIBUTION
Pressure redistribution can be achieved by removal of pressure from the affected part of the body or by reducing pressure by spreading weight more widely (Figure 6).

Independent movement
Spontaneous movement is the usual mode of pressure relief for persons with intact neurological systems. An early study found that patients who moved spontaneously fewer than 25 times each night were at significantly higher risk of pressure ulcers than those who moved more frequently. Wherever possible, patients should be encouraged to move themselves. For patients who move spontaneously, sometimes no additional repositioning is needed. Patients who are reluctant to move, due to actual or anticipated pain with movement, or because of the sedative effects of analgesia, need to be reminded to move. The impact of making small, frequent movements has been studied by testing the idea that nursing staff could move a patient slightly with each contact, eg by lifting a leg or moving an arm, to relieve pressure. The studies suggested that interface pressure was reduced under the areas moved, and in a small study a reduction in the number of pressure ulcers was observed. However, caution must be applied: unless the heels and pelvis are moved, such body movements do little to reduce pressure intensities and durations at these critical locations.

Repositioning
Repositioning should be considered for all those deemed to be at risk of pressure ulceration. More mobile patients will be able to reposition themselves (see above), but others may require assistance. Repositioning may not be suitable for all patients: some patients in a critical condition may be destabilised by repositioning. However, this is not always the case even in patients in poor haemodynamic condition. Therefore, the decision to reposition a critically ill patient should be individualised.

Frequency of repositioning
A systematic review of pressure ulcer prevention strategies found insufficient evidence to support a specific repositioning regimen. The frequency of repositioning should be based on the patient’s tissue tolerance, level of mobility, general medical condition and the support surface in use. The traditional 2-hourly repositioning regimen may provide a useful starting point from which frequency can be adjusted. An effective repositioning regimen will be indicated by the absence of persistent erythema over bony points.
prominences. If persistent erythema occurs, this may indicate that more frequent repositioning is required and that the current support surface is perhaps not optimal for the patient.

The use of a pressure redistributing support surface does not eliminate the need for repositioning. However, it may be possible to reduce the frequency of repositioning. In one study, for example, 4-hourly turning on a viscoelastic foam mattress was associated with lower incidence of Category/Stage II and above pressure ulcers when compared with 2- or 3-hourly turning on a standard mattress.

For patients sitting in chairs and wheelchairs, it is advised that repositioning should occur at a minimum every hour. Patients confined to wheelchairs should be taught to reposition every 15 minutes by doing ‘push-ups’ off the wheelchair or by leaning forwards.

**Positions**
For patients in bed, positions such as 90° side-lying or the semi-recumbent position are best avoided because these increase pressure over the trochanteric or sacral bony prominences respectively. Patients who must have some head of bed elevation, eg because of dyspnoea or to prevent aspiration during tube feeding, should be repositioned more frequently.

The 30° tilted side-lying position is a method of placing a patient so that they are tilted 30° along their vertical axis from the supine position. This position does not suit all patients, but may be a useful alternative for some.

Wheelchair dependent patients may benefit from a tilting wheelchair that helps to offload pressure from the ischial tuberosities.

**PRESSURE REDISTRIBUTING SUPPORT SURFACES**
Pressure redistributing support surfaces are available in several forms, eg overlays, mattresses and integrated bed systems.

An overlay is a support surface device placed on top of an existing mattress. These devices may elevate the sleeping surface to the level of the side rails and so the risk of the patient falling out of bed must be evaluated. Ideally, the bedrail should be at least 10cm (4 inches) higher than the surface of the mattress.

Pressure redistributing mattresses can often be used to replace standard mattresses, allowing for continued use of the existing bed frame.

An integrated bed system combines a bed frame and a support surface (usually an alternating pressure mattress). They are most often used for extremely high risk patients, for the treatment of pressure ulcers, and for patients who have had surgical reconstruction of pressure ulcers with flaps.

**REACTIVE SUPPORT SURFACES**
Two important principles of the mode of pressure redistribution of reactive support surfaces are immersion and envelopment.

Immersion refers to the ability of a support surface to allow a patient to sink into it (Figure 7). As the body sinks in, more of the body comes into contact with the support surface, redistributing the patient’s weight over a larger area and reducing pressure.
Immersion is greater on softer surfaces and also has the potential to be higher on thicker surfaces. However, if a material is too soft, the patient may ‘bottom out’ (ie end up sitting or lying on the underlying structure of the bed or chair because the support surface has become so compressed).

Envelopment refers to how well a support surface moulds to body contours and accommodates irregular areas (such as folds in clothing or bedding)29 (Figure 7).

Recent research has indicated that the degree of immersion and envelopment of a support surface can be impaired by increased tension at the surface of the support, especially when combined with sagging of the support surface itself38. For example, a tight cover over a mattress or seat cushion can create a hammock effect that prevents the support surface moulding to contours and produces high pressures over a small area (Figure 8).

Immersion and envelopment have important implications for patient mobility and independence. For example, it requires relatively little effort to stand from sitting or lying on wood (which has no immersion and envelopment), but the same manoeuvre from water requires more effort because of the high degree of immersion and envelopment.

FACT FILE

- Active support surfaces – also known as alternating pressure systems – redistribute pressure mainly through the inflation and deflation of sections of the support surface.
- The precise indications for and relative efficacy of the different types and models of pressure redistributing support surfaces in reducing the incidence of pressure ulcers remains under investigation.

Foam

Basic foam mattresses have become common as the standard mattress for patients in hospitals and long-term care facilities. Higher specification foam mattresses (eg those composed of layers of different densities of foam, or of viscoelastic foam) are recommended to reduce the incidence of pressure ulcers in persons at risk39.

Foam degrades and loses its stiffness over time, thereby losing its ability to conform. When a foam mattress wears out the patient may ‘bottom out’. The life span of any support surface is influenced by number of hours of use and the weight applied; a surface used by thin persons will outlive one used by bariatric patients.

Air or gel filled

Air or gel filled support surfaces comprise air or gel filled columns or compartments. The degree of immersion and envelopment provided depends upon the pressure of the air or gel in the compartments, the depth of the compartments, and the ‘give’ of the surface.

Air filled support surfaces are sometimes referred to as low air loss surfaces. However, strictly speaking, low air loss relates to a property of some support surfaces that allows air to escape from the cushions to aid management of skin temperature and moisture (see: Microclimate in context20, pages 19-25).

Air fluidised

Air fluidised support surfaces provide the greatest immersion and envelopment of any support surface. Almost two-thirds of the body can be immersed. An air fluidised support surface comprises silicone or glass beads that have pressurised air forced between them. This makes the beads take on characteristics of a fluid.

Several randomised controlled studies have shown that healing outcomes for patients with Category/Stage III and IV pressure ulcers who are managed on air fluidised support surfaces are improved in comparison with standard beds, and foam and other non-fluidised support surfaces40-43.

ACTIVE SUPPORT SURFACE – ALTERNATING PRESSURE

Alternating pressure support surfaces redistribute pressure by cyclically inflating and deflating zones of the surface (Figure 9). As a result they are less reliant than reactive surfaces on the properties...
of immersion and envelopment to redistribute pressure. The ideal frequency, duration, amplitude and rate of inflation and deflation have not been determined. A draft consensus document has recently proposed a standardised method for evaluating active support surfaces.

Iglesias et al reported that alternating pressure mattresses were likely to be more cost effective than alternating pressure overlays. In addition, the mean time to develop a pressure ulcer was more than 10 days longer on the alternating pressure mattress than on the alternating pressure overlay. Vanderwee et al found no significant difference in the incidence of pressure ulcers. There was a tendency for more sacral pressure ulcers in patients on alternating pressure mattresses in patients who were identified as being in need of preventive measures based on the Braden scale.

A literature review of 15 randomised controlled trials concluded that when taking into account methodological issues, alternating pressure mattresses are likely to be more effective than standard hospital mattresses in the prevention of pressure ulcers.

Support surface selection
Selection of a suitable support surface for pressure redistribution (Table 1) should not be based on risk assessment score alone, but should also take into consideration:

- level of mobility within the bed – ie how much the patient can move when in bed and whether they are able or need to be able to get themselves out of bed
FACT FILE

Regular observation is essential in evaluating the efficacy of pressure redistribution strategies. Any sign of pressure damage should prompt re-evaluation of the strategies in place.

- Patient comfort – Some patients find some support surfaces uncomfortable.
- Need for microclimate management – Some support surfaces assist with managing heat and moisture directly below the patient (see: Microclimate in Context25, pages 19–25).
- Care setting – For example, some integrated bed systems are unsuitable for home settings because of their weight and the need for an alternative power source, e.g., a generator, in case of loss of electrical power.

Even so, one study has shown that reimbursement guidelines, not patient condition, were most clearly associated with support surface selection46.

Higher specification foam mattresses (e.g., viscoelastic foam mattresses) are suitable for many at-risk patients, but those at higher risk will need a powered support surface that is able to change its load distribution properties.

Bariatric patients may be too heavy for some pressure redistributing support surfaces and require versions with extra width or features designed to accommodate high patient weight.

Additional features of integrated bed systems may include lateral rotation or vibration of the support surface to assist patients who have problems with ventilation and perfusion. ‘Turn assist’ is designed to aid repositioning, examinations and linen changes; it is not intended for patients to use in turning themselves.

OBSERVATION AND RE-EVALUATION

Once pressure redistribution strategies have been set in place, it is important to assess their effectiveness. The most important indicator is the presence or absence of changes in skin status, especially over the bony prominences. If there are indications of pressure damage, the prevention strategies may need to be intensified and/or modified. Changes in the condition of patients and their ongoing risk levels should also be monitored as these may alter the prevention strategies required.

When a specialised support surface is in use, carers should check regularly that the device is working properly and ensure that:

- A foam mattress is still ‘springing back’ to its original position when pressure is removed.
- Air-filled devices are properly inflated.

- Gel mattresses have gel throughout them and that there are no areas where gel has been moved away.
- An alternating air mattress is inflating and deflating properly.
- A powered device is plugged into a power supply.

All support surfaces, hospital beds and integrated bed systems have a finite term of use, but the exact lifespan is currently unknown. Healthcare practitioners need to be mindful of this and when pressure ulcers fail to heal consider whether a ‘worn out’ support surface may be the cause or play a role.

CONCLUSION

In addition to a direct effect, pressure also acts indirectly through the generation of shear stresses to produce pressure ulcers. The ability of pressure to produce pressure damage in soft tissues is related to the intensity and duration of the applied pressure. Patients who are unable to move or ask to be moved are those most at risk from pressure. Interventions to reduce the effect of pressure and reduce the incidence of pressure ulcers include patient repositioning and the use of specialised support surfaces.

Decisions on which support surface to use can be enhanced through appreciation of how surfaces work and for which patients each device is most suitable. However, despite expert clinical opinion, the choice of support surface is often made on a financial basis. Continued research into the effectiveness of pressure redistributing support systems in reducing the incidence of pressure ulcers will guide educational priorities, aid decision making and help to secure funding for appropriate surfaces, regardless of care setting.

REFERENCES

INTRODUCTION
Shear and friction are often mentioned alongside pressure in the context of pressure ulcers. For example, the most recent definition of pressure ulcers, produced by an international collaboration of the National Pressure Ulcer Advisory Panel (NPUAP) and European Pressure Ulcer Advisory Panel (EPUAP), emphasises the role of pressure and states that shear can be involved in combination with pressure in the development of pressure ulcers. The same collaboration also cites shear in the context of deep tissue injury, which is defined as “due to damage to underlying soft tissues from pressure and/or shear”.

Although disputed as a direct cause of pressure ulcers, friction is considered in this paper because of its close association with shear. Pressure and shear are also intimately linked: pressure on soft tissues, especially when over a bony prominence, will cause some degree of shear through tissue distortion.

The first part of this paper clearly defines shear and friction and discusses the role of each in pressure ulcer development. The second part of the paper examines how to recognise patients at risk of skin and soft tissue injury due to shear and friction. It then discusses the actions that can be taken to avoid or minimise shear and friction and so complement other measures to reduce the overall risk of pressure ulcer development.

DEFINITIONS
The terminology surrounding shear can be confusing: ‘shear’ is often used to abbreviate the different terms ‘shear stress’ and ‘shear force’. In addition, shear and friction are often mentioned together in the context of pressure ulcer aetiology, and sometimes, inaccurately, the terms are used interchangeably.

What is shear?
Shear stress results from the application of a force parallel (tangential) to the surface of an object while the base of the object stays stationary. (Note: Pressure is the result of a force that is applied perpendicular (at a right angle) to the surface of an object (see: Pressure in context, pages 2–10).) Shear stress causes the object to change shape (deform) (Figure 1). The amount of deformation caused by shear stress is quantified as shear strain.

In common with pressure, shear stress is calculated in terms of the force applied over the area to which it is applied (Box 1) (see: page 14 and Box 2 for more detail). Shear stress is expressed in the same units as pressure: most commonly as pascals (Pa), or sometimes as newtons/square metre (N/m²).

What is friction?
Friction is defined as the force that resists the relative motion of two objects that are touching, and is measured in newtons (N). However, the term ‘friction’ is also frequently used to mean the action of one object rubbing against the other (see: page 14 and Box 2 for more detail).

WHAT CAUSES SHEAR STRESSES?
Gravity produces a force that pulls a patient onto the surface they are resting on. The opposing force produced by the surface can be divided into two components:

- a perpendicular component – which results in pressure
- a tangential component – which results in shear stresses (Figure 2, page 12).

BOX 1 Defining shear stress

<table>
<thead>
<tr>
<th>Shear stress =</th>
<th>Tangential force applied (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(pascals or N/m²)</td>
<td>Area of application of force (m²)</td>
</tr>
<tr>
<td>1Pa = 1N/m²</td>
<td>1kPa = 1000N/m²</td>
</tr>
</tbody>
</table>

Definitions of shear stress:

- “An action or stress resulting from applied forces which causes or tends to cause two contiguous internal parts of the body to deform in the transverse plane (ie shear strain).”
- “The force per unit area exerted parallel to the plane of interest.”
Friction contributes to the development of shear stresses by tending to keep the skin in place against the support surface while the rest of the patient’s body moves towards the foot of the bed or the edge of the seat. The relative movement of the skin and underlying tissues causes shear stresses to develop in the soft tissues overlying bony prominences such as the sacrum.

The angle of the back support of a bed, or the angle of the backrest of a seat or wheelchair, strongly influences the level of shear stresses in tissues. All angles between an erect sitting posture and horizontal lying will cause shear stresses due to the body’s tendency to slide downwards along the slope. Lying with the backrest at an angle of 45° will cause a particularly high combination of shear stresses and pressure at the buttocks and sacral area because, in this posture, the weight of the upper body divides equally into perpendicular and tangential forces.

Shear stresses in tissues may also be caused by localised pressure applied to a skin surface. The application of pressure causes compression of the tissues, and by doing so distorts adjacent tissues (Figure 3). This is sometimes known as pinch shear. Steep pressure gradients, ie large changes in pressure across a small surface area, are likely to produce high pinch shear.

Shear stresses are thought to act in conjunction with pressure to produce the damage and ischaemia of the skin and deeper tissues that results in pressure ulcers. The mechanisms involved include distortion of tissues, pinching and occlusion of capillaries crossing tissue planes, reductions in blood flow, and physical disruption of tissues or blood vessels.

### Tissue distortion

In layered objects, eg body tissues, shear stresses can cause one layer to move relative to another (Figure 4). When shear stresses are applied to tissues, the amount of movement between the layers in the tissues – ie the degree of potential for producing blood vessel occlusion and physical disruption of tissues – is affected by the looseness of the connective tissue fibres between the layers and the relative stiffnesses of the tissue layers.

In aged skin, skin elasticity and skin turgor tend to be reduced. As a result, more pronounced skin tissue displacements can take place in skin and subdermal layers when external forces are applied.

Differences in the stiffnesses of distinct tissue layers mean that they deform to varying extents when an external force is applied. Stiffer tissues deform to a lesser extent than materials of lower stiffness. Table 1 shows that the greatest difference in stiffness of adjacent tissues, ie the greatest potential for shear stresses to occur, is between the bone and muscle, but that
potential for shear stresses also occurs between muscle and adipose tissue, and between adipose tissue and skin.

This helps to explain why pressure ulcers frequently develop over bony prominences, where interface pressures also tend to be highest. Patients with prominent bones are particularly prone to shear stresses and pressure, and slender body types tend to have higher shear stresses and pressure at the coccyx and sacrum than do obese body types.

Effects on blood vessels
Shear stresses can reduce or prevent blood flow through a number of mechanisms:
- Direct compression and occlusion of blood vessels (Figure 4)
- Stretching and narrowing of dermal capillary beds – when sufficiently high shear stresses are applied, the internal diameter of the capillaries becomes inadequate for blood flow
- Bending and pinching of blood vessels running perpendicular to the skin surface.

The capillaries in adipose tissue are also vulnerable to the effects of shear stresses because adipose tissue lacks significant tensile strength (ie it distorts and tears apart easily).

Deeper and larger blood vessels may also be affected by shear stresses. The blood supply for skin and subcutaneous tissues can be traced back to arteries that arise below the deep fascia and muscle. These arteries – known as perforator vessels – tend to run up perpendicular to the surface and to supply considerable areas. Their perpendicular route makes them particularly prone to shear stresses, and may explain the observation that some larger sacral pressure ulcers tend to follow the supply pattern of specific blood vessels.

Pressure and shear stresses usually work in tandem to reduce blood flow. Biomechanical modelling has demonstrated that shear stresses applied in addition to pressure cause greater obstruction and distortion of capillaries in skeletal muscle around bony prominences than does pressure alone. At sufficiently high levels of shear stresses, only half as much pressure is required to produce blood vessel occlusion as when little shear stress is present. Conversely, if shear stresses are reduced, tissues can tolerate higher pressures without blood flow occlusion.

Measuring shear stresses
Several devices are available for measuring shear stresses at skin surface interfaces; some devices also simultaneously measure interface pressure. Internal shear stresses are difficult to measure directly, but have been estimated using computer modelling and using computer modelling in combination with magnetic resonance imaging (MRI).

WHAT AFFECTS FRICTION?
Friction force at the patient-support surface interface is dependent on the perpendicular force and the coefficient of friction of the skin.

| Table 1: Relative stiffness of body tissues (based on animal studies) |
|--------------------------|------------------|-------------------|
| Body tissue              | Stiffness (as indicated by elastic modulus (kPa)) |
| Bone                     | 20,000,000       |
| Muscle                   | 7                |
| Adipose tissue           | 0.3              |
| Skin                     | 2–5              |

Figure 4: Effect of shear stress on body tissue layers (courtesy of T Ohura)
and the contact surface (Box 2). The higher the perpendicular force, the higher the friction force. Similarly, the higher the coefficient of friction, the higher the friction force and the greater the force required to make the patient move in relation to the support surface.

The coefficient of friction of textiles or other materials against skin is mainly influenced by:
- the nature of the textile – eg rougher textiles produce higher coefficients of friction
- skin moisture content and surface wetness – these increase the coefficient of friction and are particularly relevant in the clinical context where skin may be damp from perspiration or because of incontinence (see: Microclimate in context, pages 19–25)
- ambient humidity – high ambient humidity may increase skin moisture content or induce sweating and therefore increase coefficient of friction (see above).

A study looking at the interaction between skin and a polyester/cotton textile confirmed that as skin moisture increased, the coefficient of friction also increased. The same study found that the coefficient of friction for wet fabric on skin was more than double the value for dry fabric on skin.

The significance of friction in the context of pressure ulcer development lies mainly in its contribution to the production of shear stresses. When the tangential force applied by friction at the skin surface is larger than the perpendicular force (pressure), or when a small amount of pressure with a large tangential force is applied to the skin, abrasions, superficial ulceration or blistering may occur. If the skin is already irritated or inflamed, eg by maceration, incontinence-associated dermatitis or infection, superficial damage due to friction will occur more easily. Friction applied to the skin surface can also cause shear stresses in deeper tissue layers such as muscle.

HOW MIGHT FRICTION CONTRIBUTE TO PRESSURE ULCER DEVELOPMENT?

The magnitude of friction force is dependent on the perpendicular force and a characteristic of the interaction of the two objects known as the coefficient of friction. Moist skin has a higher coefficient of friction than dry skin, and is therefore more likely to be exposed to higher levels of friction and shear stresses.

Much research is required to fully unravel how shear stresses cause tissue damage, the effect of the frequency and/or speed of postural changes on shear stresses, and which patients are at greatest risk of injury from shear stresses. Many of the interventions aimed at reducing shear stresses and friction revolve around attempts by healthcare professionals, carers or patients themselves to move or reposition patients, as it is during such manoeuvres that there is increased risk of shear stress and friction occurring.

BOX 2 Friction

Friction force opposes externally applied forces; movement of one surface against another will only occur when the applied force is greater than the friction force. The friction force produced by two surfaces in contact is dependent on the perpendicular force (related to the weight of the object) and the coefficient of friction. The coefficient of friction is a value that is dependent on the properties of the two objects that are in contact.

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Measuring friction

Experiments related to the measurement of friction usually determine the coefficient of friction of the materials being examined. A standardised method used commonly calculates the coefficient of friction between a block of metal and a fabric. This standardisation should allow for comparison between textiles to be made easily. However, differences in equipment and methods of measurement used in those studies that have been conducted make comparisons of results difficult, and the role of textiles in the prevention and formation of pressure ulcers is understudied.

MANAGEMENT OF SHEAR STRESSES AND FRICTION

Alongside pressure redistribution, patient repositioning and mobilisation, strategies to reduce shear stresses and friction form an important part of best clinical practice to reduce patients’ overall risk of pressure ulcer development.

A number of guidelines for the prevention of pressure ulcers have developed recommendations to assist with decision making about appropriate health care. These include the recent guidelines produced by the NPUAP and EPUAP and those produced by the Registered Nurses’ Association of Ontario. Decisions for care will require clinical judgement based on
patient comfort and wishes, and other treatment or care needs.

The principles involved in minimising the effects of shear stresses and friction include:

- **decreasing tangential forces** – eg during lying, by minimising head of bed elevation, and during sitting, by avoiding sliding downwards/forwards.
- **avoiding actions that induce tissue distortion** – eg avoiding sliding or dragging, by ensuring that patients are positioned in a way that does not allow them to slip easily and by ensuring that body tissues are not dragged upon during repositioning or left distorted following repositioning.
- **increasing contact area with support surfaces** – this spreads the perpendicular and tangential loads and friction force over a larger area, reducing the localised pressure and shear stresses.

The use of lower coefficient of friction textiles to cover support surfaces will reduce friction force and shear stresses. However, a balance is required: if the coefficient of friction is too low, the patient may slide around on the support surface and be difficult to place in a stable position.

**Clinical practice steps**

Best clinical practice begins with identification of those at risk and ends with an evaluation of the impact of implementation, ie effect on incidence and prevalence of pressure ulcers. Clinical recommendations from the recent NPUAP and EPUAP guidelines that particularly relate to shear stress and friction are reviewed in the practice steps below. The majority of these recommendations are classified as having ‘strength of evidence = C’, meaning that they are supported by indirect evidence and/or expert opinion.

**Step 1: Identify those at risk from shear stresses and friction**

- Establish a risk assessment policy in all health care settings.
- Consider the potential impact of following factors on an individual’s risk of pressure ulcer development: friction and shear, sensory perception, general health status and body temperature.

**Box 3 Patients at risk of shear stresses and friction**

Patients that:

- must have head of bed elevation because of difficulty breathing or the use of medical devices such as ventilators or tube feeding equipment
- are difficult to reposition without some sliding across bed sheets or support surface
- slip or slide from a position that they have been placed in when in a bed, chair or wheelchair – eg patients who are unable to or find it difficult to position themselves because they are immobile, have sensory loss or are physiologically unstable
- are too weak or too unstable to be able to reposition themselves effectively without dragging across sheets or support surfaces
- have moist, wet or macerated skin where the skin touches a support surface or another skin surface (skin folds/pannus) – eg due to sweat, incontinence or leaking dressings
- are exposed to high pressures, especially over bony prominences – eg very thin patients
- are obese – risk may be increased because of immobility and difficulties with transfers or repositioning, increased sweating and poor perfusion of adipose tissue
- have decreased skin elasticity and/or turgor – eg due to ageing or dehydration
- have fragile skin – eg due to steroid or anticoagulant use, scar tissue over a healed pressure ulcer, inflammation or oedema
- have signs of existing skin friction damage – eg superficial abrasions or blistersing on areas in contact with support surfaces (Figure 5)
- have a current or healed pressure ulcer
- have developed undermining in an existing pressure ulcer – this may signify that shear stresses are being applied; the undermining in such cases will be towards the underlying bony prominence
- have an irregularly shaped pressure ulcer
- tend to rub their heels on the bed due to agitation – eg as a result of pain or dementia
- have dressings that show partial peeling along one edge – the forces involved may be coming from the side of the peeling.
The three most commonly used pressure ulcer risk scales (Braden, Norton and Waterlow) all recognise moisture or incontinence as a risk factor for pressure ulcers. However, only the Braden scale specifically evaluates friction and shear; it does so on the basis of the level of assistance required to move, frequency of sliding in a bed or chair, and the presence of spasticity, contractures or agitation that cause friction.

**Step 2: Assess those at risk from shear stresses and friction**

- Ensure that a complete skin assessment is part of the risk assessment screening policy in place in all health care settings.

Complete skin assessment will enable clinicians to determine the presence of existing pressure ulcers and to look for signs that indicate that the patient is at risk of shear stresses and friction (see Step 1).

Although it is very important to distinguish clinically between pressure ulcers and moisture lesions such as incontinence-associated dermatitis, the presence of moisture lesions increases the coefficient of friction of skin, and consequently risk from shear stresses and friction.

If damage due to shear stresses and friction has already occurred, determining how shear and friction were involved may suggest interventions to prevent further damage. For example, if a wheelchair user develops damage, analysis of how transfers are made may reveal that ‘drag’ is occurring and suggest interventions that reduce drag.

**Step 3: Provide care for those at risk from shear stresses and friction**

### Skin care

- Do not vigorously rub skin that is at risk for pressure ulceration.

Skin rubbing is an outdated practice that unfortunately persists in some places. When clinicians rub already reddened and inflamed tissues there is a possibility of damage to the underlying blood vessels and/or to the fragile skin. If emollients are applied to skin, they should be applied gently to avoid unnecessary trauma. Incompletely absorbed emollients that leave a sticky residue on the skin and may increase the coefficient of friction should be avoided. There is anecdotal evidence that application of silicone-based lotions to the skin of patients who have a lot of drag or resistance during repositioning may ease friction.

Management of skin moisture to avoid it becoming damp or macerated is important to avoid increasing the coefficient of friction of the skin (see: Microclimate in context, pages 19–25).

- Consider using film dressings to protect body areas at risk for friction injury or risk of injury from tape.

An increasing range of dressing products (including film dressings) that aim to reduce shear stresses and friction over vulnerable areas is under investigation. Transparent dressings, eg films, aid monitoring of the underlying skin. A study using an animal model found that film dressings produced greater reductions in shear and pressure than did other types of dressings.

Dressing types that have been studied clinically include a hydrocolloid dressing that has a low coefficient of friction outer surface. This dressing was found to reduce shear force when applied to areas susceptible to shear damage such as the heel, and to significantly reduce the incidence of persistent erythema when placed over the greater trochanter. In a more recent study, application of a soft silicone dressing to the sacrum in high risk intensive care patients was associated with a reduction in sacral pressure ulcer incidence to zero.

### Positioning

- Select a posture that is acceptable for the individual and minimizes the pressure and shear exerted on the skin and soft tissues.

Limit head-of-bed elevation to 30 degrees for an individual on bedrest, unless contraindicated by medical condition. Encourage individuals to sleep in a 30 to 40 degree side lying position or flat in bed if not contraindicated.

- Use transfer aids to restrict friction and shear. Lift – don’t drag – the individual when repositioning.

- If sitting in bed is necessary, avoid head of bed elevation or a slouched position that places pressure and shear on the sacrum and coccyx.
The input of specialist advisers on seating and support surfaces may be required to ensure that the patient is placed in a comfortable position that minimises shear and friction, and avoids head of bed elevation. Slight knee gatch (elevation of a bend in a support surface at the level of the knees) engagement may help to prevent the patient from sliding down the bed.

The recommendation for limiting head of bed elevation is based on a study performed in healthy volunteers. This found that the 30° semi-Fowler position (which involves 30° head of bed elevation and 30° elevation of the legs) produced lower pressure and shear stresses than did a supine position with 30° head of bed elevation50. The same study found that a 30° side-lying position gave lower interface pressure readings than the 90° side-lying position51. However, patient positioning needs to consider all of the patient’s needs. For example, if the patient is being ventilated, critical care protocols may recommend 30-45° head of bed elevation.

The risk of friction burns can be reduced by careful repositioning of patients to avoid dragging across the support surface cover, and the use of turning sheets or transfer aids51. Even so, a clear understanding of how shear stresses and friction occur will undoubtedly assist clinicians in consistent implementation of aspects of pressure ulcer prevention protocols designed to minimise shear stresses and avoid increasing the coefficient of friction of skin.

**CONCLUSION**

Shear stresses – and by association, friction – are important extrinsic factors involved in the development, and sometimes persistence, of pressure ulcers. However, many uncertainties remain about the role and critical levels for shear stress and friction in pressure ulcer development. Even so, a clear understanding of how shear stresses and friction occur will undoubtedly assist clinicians in consistent implementation of aspects of pressure ulcer prevention protocols designed to minimise shear stresses and avoid increasing the coefficient of friction of skin.

**REFERENCES**

8. Linder-Ganz E, Gefen A. Mechanical compression-induced


42. Waterlow J. The Waterlow score card. Available at: www.waterlow.co.uk/the-waterlow-score-card.html.


INTRODUCTION
Numerous factors have been implicated in the aetiology and pathophysiology of pressure ulcers1-3. Even so, it is clear that there is much still to be learned about the complex interactions between the many intrinsic and extrinsic factors involved. Recently, interest has been building in how modifying the environment at or near the skin surface – the microclimate – may affect risk of pressure ulcer development4,5.

This paper defines the main parameters involved in current understanding of microclimate and explores what is known about the relationship between microclimate and pressure ulcer development. It also describes interventions that may beneficially alter microclimate. Discussion includes the effect of different support surfaces on microclimate and how management of microclimate may help to avoid pressure ulcers.

WHAT IS MICROCLIMATE?
In 1976, Roaf reported that the first UK-based conference on pressure ulcers highlighted known contributory factors to pressure ulcer development: “We know how to avoid bed sores and tissue necrosis – maintain the circulation, avoid long continued pressure, abrasions, extremes of heat and cold, maintain a favourable micro-climate, avoid irritating fluids and infection”6.

The maintenance of a favourable microclimate was seen in early pressure ulcer publications to be a key modifier of the ability of skin and underlying soft tissue to withstand prolonged stress, ie pressure and shear. The important role of microclimate modification in the prevention of pressure ulcers has largely been overlooked since the 1970s, but is now regaining attention.

Microclimate was suggested by Roaf to include skin temperature, humidity and air movement6. However, today, the use of the term microclimate in relation to pressure ulcers usually refers to:

- skin surface or tissue temperature
- humidity or skin surface moisture at the body-support surface interface2,7.

As described later, some support surfaces use air movement to influence temperature and humidity/moisture at the interface between skin and the support surface.

Studies examining the effects of elements of microclimate on skin and pressure ulcer development are inconsistent in the definitions used, making study interpretation and comparison difficult. Some of the definitions that have been used are discussed below.

Skin surface temperature
Methods used to measure skin surface temperature include:

- measurement at ‘radiative equilibrium’ – ie when the temperature of exposed skin has reached steady state following exposure to air
- measurement at the skin–support surface interface with the patient still in contact with the surface or very soon after moving out of contact with the surface.

The first method – measurement at radiative equilibrium – provides an indication of the ‘intrinsic’ skin (but not core body) temperature of the patient (although still subject to external variables such as ambient temperature). The second method provides an indication of the temperature at the skin–support surface interface.

Temperature may be measured directly, eg with a thermometer, or indirectly by using infrared thermal imaging (thermography)8.

Humidity
Within the literature relating to microclimate, ‘humidity’ and ‘skin moisture’ are sometimes used synonymously. However, strictly speaking humidity relates to the amount of water vapour in the air:

- absolute humidity - is expressed as the weight of water in grams per cubic metre of air (g/m³)

- relative humidity (often abbreviated to humidity) - is a ratio expressed as a percentage that relates the amount of water vapour in the air at a specific air temperature to the maximum amount of water vapour that body of air would hold at that temperature (Box 1). The relative humidity of the general surroundings is known as ambient humidity.
Absolute humidity is affected by air temperature: warmer air is able to hold more water vapour than cooler air. Therefore, for the same absolute humidity, warmer air will have a lower relative humidity than cooler air.

Relative humidity is measured using a device known as a hygrometer. In relation to skin, relative humidity may be measured at the interface between the skin and a support surface, or just above exposed skin.

### Skin moisture

Skin moisture is difficult to define: it may refer to the presence of fluid on the skin surface (‘wetness’) from perspiration, incontinence or wound/fistula drainage, or to the moisture content of the outermost layer of the skin itself (the stratum corneum).

Skin moisture can be assessed subjectively, eg using the moisture subscale of the Braden scale for assessing risk of pressure ulceration; this classifies skin as dry or having some degree of moisture as indicated by frequency of linen changes and detection of dampness on the skin surface. Methods to quantitatively assess the moisture content of the stratum corneum include measurement of electrical properties such as skin surface conductance or capacitance.

### Air movement

Air movement is the least investigated factor of Roaf’s original definition of microclimate, but is used by some support surfaces to aid microclimate control by modifying temperature and humidity/skin moisture. The flow of air can be expressed quantitatively as the velocity (speed) of airflow across the skin, eg in metres per second, or by the rate at which air is pumped through a support surface, eg in litres per minute.

### BOX 1 Definitions of humidity

<table>
<thead>
<tr>
<th>Definition</th>
<th>Formula</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absolute humidity (g/m³)</td>
<td>weight of water vapour / volume of air</td>
</tr>
<tr>
<td>Relative humidity at a specific temperature (%)</td>
<td>amount of water vapour in the air at a specific temperature / maximum amount of water vapour that can be held in the air at that temperature</td>
</tr>
</tbody>
</table>

### HOW IS SKIN SURFACE TEMPERATURE RELATED TO PRESSURE ULCER RISK?

Raised body temperature (pyrexia) is a recognised risk factor for pressure ulcers. It is well established that increasing body temperature by 1°C raises the metabolic activity (ie the need for oxygen and energy) of body tissues by about 10%. By definition, ischaemia occurs when tissue perfusion is not sufficient to meet the needs of the tissue. Therefore, when metabolic needs are raised, a smaller reduction in tissue perfusion will produce ischaemia than when metabolic needs are steady. This suggests that in a patient with elevated body temperature and compromised tissue perfusion due to exposure to pressure and shear, ischaemia and tissue damage may occur more quickly and at lower levels and/or shorter durations of pressure/shear than if body temperature was normal.

This concept has been extended to suggest that increased skin temperature may play a role in the development of pressure ulcers.

In addition, temperature affects the strength of the stratum corneum: at 35°C the mechanical strength of the stratum corneum is 25% of that at 30°C. In contrast, low core body temperature during surgery is associated with pressure ulcer development. To examine whether preventing hypothermia during surgery would reduce the incidence of pressure ulcers, Scott et al provided forced air warming therapy during surgery to 338 patients. There was an absolute risk reduction in pressure ulcer incidence of 4.8% and a relative risk reduction of 46% between those who received warming and those who received standard care. However, this difference did not achieve statistical significance.

### What affects skin temperature?

It seems intuitive that raised core body temperature would correlate with increased skin temperature, and that this would perhaps help to explain why pyrexia is a risk factor for pressure ulcers. However, clear evidence of a correlation is lacking and one small study found a negative correlation between these parameters.

Other factors that may increase skin temperature include high ambient temperature, high ambient humidity, low exposure to air, and contact with another surface (eg clothing, support surfaces, dressings and incontinence pads).
The skin has a major role in regulating body temperature. The two main mechanisms involved in cooling are:

- dermal vasodilatation – which increases skin blood flow and causes heat loss by convection and conduction
- sweating – perspiration cools the skin through evaporation.

These responses may be triggered by increases in core body temperature, e.g. during pyrexial illness or exertion, increases in ambient temperature, or covering the skin with clothing or a support surface. Local perspiration increases considerably when the skin is warmed above about 33°C. Perspiration may also be triggered in conditions such as shock, hyperthyroidism, and hypoglycaemia. When ambient humidity is high, evaporation of perspiration may be slowed causing perspiration to accumulate on the skin.

Increased perspiration is particularly relevant to pressure ulcer risk because moisture on the skin surface can reduce the skin’s resilience and increase the coefficient of friction of skin, making it more prone to pressure, shear stresses and friction (see: Pressure in context21, pages 2-10 and Shear and friction in context22, pages 11-18).

Conversely, the skin’s contribution to conservation of body heat is largely mediated by dermal vasoconstriction.

What is normal skin temperature?

Surprisingly, there is limited information on normal skin temperature over anatomical sites prone to pressure ulcer development. One key study proposed that sites prone to pressure ulcer development were cooler than adjacent body sites. However, there were several weaknesses in this study; for example, ambient room temperature was unreported, as was the duration for which the skin was exposed to room temperature prior to temperature measurement.

Can skin surface temperature predict pressure ulcer development?

Several studies have investigated skin temperature associated with early (Category/Stage 1) pressure damage. These found inconsistent changes in skin temperature of pressure damaged areas: it could be increased (possibly as a result of inflammation), the same, or decreased (possibly as a result of ischaemia) in comparison with temperature at undamaged areas.

The few studies that have examined whether skin temperature can predict pressure ulceration have been inconclusive. In a prospective cohort study, Clark measured the sacral skin temperature of 52 elderly hospital patients. Of this cohort, six developed pressure ulcers; however, the skin temperature of the patients who did and did not develop pressure ulcers was similar. This study was confounded by a non-uniform allocation of support surfaces to the recruited subjects. However, one small study of neurologically impaired patients found that sacral skin temperature may increase 24–96 hours before sacral pressure ulcer development by at least 1.2°C.

A recent study has examined whether skin temperature regulation may predict pressure ulcer development. A small sample of nursing home patients wore skin temperature monitors taped to the right mid-axillary line continuously for five days. The study found that patients at high risk or who went on to develop pressure ulcers had least variability in skin temperature, suggesting impaired skin temperature regulation. However, it is unknown whether reduced ability to regulate skin temperature is directly related to pressure ulcer development or if it is a general marker of declining physiological condition.

How does changing local skin temperature affect risk of pressure ulcer development?

Some investigators have examined how changing local skin temperature affects the impact of pressure on tissues. In an animal study, a known pressure (100mmHg) was applied for five hours with indentors heated to 25, 35, 40 or 45°C. Moderate muscle damage was seen at 35°C and cutaneous and deep tissue damage were observed at 40 and 45°C (note that there may have been an element of thermal damage at 45°C). No cutaneous or muscle damage was observed where load was applied at 25°C, suggesting that local cooling may have a protective effect.

More recently, Lachenbruch has argued from prior studies (including that of Kokate et al.) that a 5°C reduction in skin–support surface interface temperature would confer tissue protective effects similar in magnitude to the interface pressure reductions afforded by the most expensive support surfaces. This hypothesis remains untested.
Although cooling may provide some protective effects, as mentioned previously hypothermia during surgery may contribute to the development of postoperative pressure ulcers.

**HOW DO HUMIDITY AND SKIN MOISTURE RELATE TO PRESSURE ULCER RISK?**

Increased skin moisture, especially when due to incontinence, has long been recognised as an important risk factor for pressure ulcer development. However, there is limited quantitative data on skin moisture or humidity to support this view.

Clark reported that the humidity just above the sacral skin of elderly hospital patients who subsequently developed Category/Stage II pressure ulcers was higher than the humidity of patients who did not.

Increased skin moisture as measured by electrical capacitance has been found in a study in Indonesia and in a small pilot study to correlate with the development of pressure ulcers. However, further studies are required to determine whether measuring skin moisture in this way may prove useful in identifying patients who would benefit from additional intervention to prevent pressure ulceration.

**Effects of excessive skin moisture**

Excessive moisture on the skin surface, eg as a result of perspiration, urinary or faecal incontinence, wound/fistula drainage or vomit, is thought to contribute to increased risk for the development of pressure ulceration by weakening skin. Moisture can weaken the crosslinks between the collagen in the dermis and soft the stratum corneum. This can cause maceration (or incontinence-associated dermatitis) if the liquid involved is urine – Figure 1 – and also increases the exposure of underlying blood vessels to the effects of pressure and shear stresses.

In addition, excessive moisture can significantly increase the skin’s coefficient of friction, leading to increased likelihood of skin damage from friction and shear stresses (see: *Shear and friction in context*, pages 11-18).

Relative humidity also affects the strength of the stratum corneum: at a relative humidity of 100% the stratum corneum is 25 times weaker than at 50% relative humidity.

**Effects of excessive skin dryness**

Ageing skin is less resilient and more vulnerable to damage than is younger skin because it is generally thinner, structurally weaker and drier. Dry skin has reduced lipid levels, water content, tensile strength, flexibility and junctional integrity between the dermis and the epidermis. Low ambient humidity reduces water content in the stratum corneum. In the USA, the Agency for Health Care Policy and Research guidelines on the prevention of pressure ulcers, recommends avoiding ambient relative humidity below 40% to reduce the likelihood of dry skin.

**HOW DOES AIR MOVEMENT RELATE TO PRESSURE ULCER DEVELOPMENT?**

There appears to have been no research to look specifically at the possible role of air movement in the aetiology of pressure ulcers. The relevance of air movement may relate to its potential to affect skin temperature and moisture content through convection and evaporation.

**WHAT DON’T WE KNOW ABOUT MICROCLIMATE AS A CAUSE OF PRESSURE ULCERS?**

The body of literature linking pressure ulcers and microclimate is relatively small, with little characterisation of interactions between skin and fabric (eg support surface covers). Furthermore, there is evidence of wide intra- and inter-individual differences in skin microclimate parameters, but the effect of these on pressure ulcer development is unclear. Interpretation of available data is therefore challenging.

The *in vivo* experimental studies on interactions between skin and fabrics that have been performed have rarely led to any significant results or definitive conclusions. This may be partly explained by the considerable differences that may exist in skin condition (eg hydration, surface roughness, adhesion between skin layers) between individuals and between different anatomical locations in the same individual.

**CLINICAL MANAGEMENT OF MICROCLIMATE**

The needs of the patient should be carefully assessed before modifications in temperature and humidity/skin moisture are undertaken.
A key strategy in the management of microclimate is control of the underlying cause of the extreme of temperature or skin moisture, eg pyrexia or incontinence. Patients who are hot may be cooled using simple measures such as reduction in the number of covers, avoidance of plastic mattress coverings, frequent repositioning, use of a fan and wearing breathable cotton clothing. Where available in climates with high ambient humidity, air conditioning may be helpful in cooling and reducing humidity.

Repositioning

The importance of the role of repositioning in controlling microclimate should not be underestimated. Some mattress covers are made of material that prevents dissipation of heat. Repositioning patients allows skin that has been in contact with the mattress cover to be exposed to air and to become cooler. It also provides an opportunity for perspiration to evaporate.

Skin care

Barrier creams and sprays can be useful in protecting moist skin from further damage, especially from urine. However, it is best to manage incontinence where possible so that the skin does not come into contact with urine in the first place.

For patients who are incontinent and require the use of absorbent underpads, use of breathable pads which allow transmission of moisture vapour is preferable. Care is required to ensure that the use of underpads does not interfere with the pressure redistribution or microclimate management properties of any support surface in use.

Bariatric patients who are prone to excessive perspiration may benefit from frequent washing and changing of clothes and bed linen to control skin moisture. Accumulation of moisture in the skin folds of bariatric patients may be a particular problem, and result in intertriginous dermatitis with bacterial and candidal infections (Figure 2).

The use of emollients can assist in ameliorating dry skin and may reduce the risk of skin damage. They should be applied liberally and frequently (eg up to three to four times daily) when the skin is very dry, and following washing or bathing when they will help to trap water onto the skin.

Role of support surfaces in managing microclimate

Any surface that comes into contact with skin has the potential to alter microclimate by changing the rate of evaporation of moisture and the rate at which heat dissipates from the skin. The overall effect on microclimate is dependent on numerous factors, including the nature of the support surface itself (ie what is made from, how the material is conformed, what sort of cover it has).

For example, foam surfaces tend to increase skin temperature because they have poor heat transfer properties. The effect of foam products on moisture depends on the porosity of the cover. Gel filled products may initially have a cooling effect that wears off after more than two hours of patient contact and tend to increase humidity at the skin surface. Fluid filled products that utilise high heat capacity liquids have the potential to reduce skin temperature. Alternating pressure air mattresses may limit skin temperature increases.

Some specialised support surfaces for beds have features that aid active management of microclimate by allowing air to flow through their surfaces, for example low air loss features or air fluidised beds. The air flow cools skin through convection and the evaporation of moisture from the skin surface.

Low air loss surfaces

Low air loss surfaces pump air into a series of cushions and then allow the air to escape through small holes (porosities) in the cushions’ covers. The air flows along the inside of a vapour permeable cover – water vapour is drawn through by the flow of air on the other side of the cover.
vapour permeable patient contact layer drawing moisture and heat through the contact layer and away from the skin (Figure 3). Low air loss surfaces have been shown to lower skin temperature and to produce lower moisture gain than standard hospital mattresses.

Air fluidised support surfaces
Air fluidised support surfaces comprise solid silicone particles the size of sand through which air is forced. The air flow causes the particles to take on properties of a liquid. The cover over the particles is porous allowing air to escape and body fluids (eg sweat, urine) to flow through (Figure 4). Air fluidised beds are considered to be the most drying support surface; the fluid loss increases linearly with increased air flow temperature.

The air flowing through low air loss and air fluidised devices is generally warmed in the range of approximately 28–35°C and can be adjusted. This facility is undeniably useful, but has to be used with considerable care to prevent inappropriate cooling or heating.

Choosing a support surface to manage microclimate
Despite the availability of several forms of low air loss and air fluidised support systems, there is little information to guide decisions about which surface to use for which patient. Choice of support surface will be guided by clinical judgement and take into account numerous factors including the patient’s need for pressure redistribution, the patient’s size, ability to turn or move independently and body temperature, the presence of moisture on the skin or concomitant conditions such as incontinence. (See: Pressure in context, pages 2–10, for further information on pressure redistributing support surfaces.)

Whilst air loss systems may be helpful in keeping immobile patients cool and dry, it is important to recognise that patients must still be turned and repositioned on these devices.

According to patient needs, the frequency of positioning may be reduced in comparison with standard mattresses (see: Pressure in context, pages 2–10).

Recent research has highlighted the importance of minimising layers of bedding between patients and low air loss support surfaces to prevent skin temperature rises.

Impact of microclimate management on pressure ulcer prevention
Support surfaces designed to aid microclimate management also provide pressure redistribution. This complicates assessment of the impact of microclimate management on incidence of pressure ulcers, and evidence demonstrating that management of microclimate directly prevents pressure ulcers is currently lacking.

Even so, clinical studies have shown that some advanced support surfaces that affect skin temperature and moisture, eg air fluidised and low air loss surfaces, are more effective than standard foam mattresses for the treatment of pressure ulcers. In addition, there is some evidence that low air loss beds reduce the incidence of pressure ulcers in intensive care.

CONCLUSION
The concept of microclimate in relation to pressure ulcers has existed for some time. However, microclimate and its elements remain to be fully defined, and its relationship to pressure ulcer development clearly characterised. Evidence to date suggests that extremes of skin temperature and/or humidity/skin moisture appear to increase the sensitivity of skin to the damaging effects of pressure, shear stresses and friction.

This suggests, therefore, that the overall aim of microclimate management should be to avoid extremes of temperature or skin moisture and to enhance patient comfort. However, further investigation is needed to establish the effects of ‘traditional’ preventive interventions (such as repositioning) and those of support surfaces on elements of skin microclimate and pressure ulcer incidence. Low air loss support surfaces and air fluidised beds are designed to assist with microclimate management, but in the absence of evidence defining optimal skin temperature and moisture levels, clinical judgement is required for effective and safe use.

FACT FILE
- Low air loss and air fluidised support systems provide mechanisms for active microclimate control.
- In the absence of clinical evidence, choice of support surface for the management of microclimate remains reliant on clinical judgement.
- Further research is required to evaluate the effectiveness of microclimate control features in preventing pressure ulcers.
REFERENCES


